Case Report

Electrical Connections between Persistent Left Superior Vena Cava and Left Atrium during Catheter Ablation for Atrial Tachycardia

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A 74-year-old man presented with palpitation and 12-lead ECG exhibited atrial premature contraction (APC) at general check-up. Holter ECG demonstrated narrow QRS tachycardia with a rate of 160/min and more than 31,000/day atrial premature beats. The P wave morphology of atrial premature beats showed negative in II, III, aVF and biphasic in V1. Venography was performed and disclosed persistent left superior vena cava (LSVC) draining into the right atrium via the markedly dilated coronary sinus (CS). Electrogram recordings from LSVC and CS were obtained with an electrode catheter via the left subclavian vein. At the level where a ventricular potential disappeared, the intra-LSVC potentials began to show a discrete second sharp potential after local left atrial signals. Double potentials were obtained within the LSVC from the lower left atrium (LA) to the higher LA. A proximal-to-distal activation sequence of the second components was observed. The interval between the 1st and 2nd component ranged from 8 to 22 msec between the proximal LSVC and distal LSVC. The double potentials resulted in fusion at the lower part of the LSVC, indicating the presence of an electrical connection between the LSVC and lower LA.
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Key words: Left superior vena cava, Ligament of Marshall, Atrial tachycardia, Atrial fibrillation, Catheter ablation

The thoracic veins play an important role in the genesis and maintenance of atrial arrhythmias.[1] The left superior vena cava (LSVC) is the embryological precursor of the ligament of Marshall (LOM), which contains multiple electrical connections to the left atrium (LA). The electrical connection has also been implicated as an arrhythmogenic substrate of reentry in atrial tachy-arrhythmias.[2–4]

Case Report

A 74-year-old man presented with palpitation and 12-lead ECG exhibited atrial premature contraction at general check-up. Holter ECG demonstrated narrow QRS tachycardia with a rate of 160/min and more than 31,000/day atrial premature beats. He was admitted for electrophysiologic study and radiofrequency ablation for the atrial tachycardia (AT).

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After written informed consent was obtained, an electrophysiologic study was performed free of antiarrhythmic agents. The P wave morphology of atrial premature beats was negative in II, III, aVF and biphasic in V1 (**Figure 1**). Venography was performed and disclosed persistent LSVC draining into the right atrium via the markedly dilated coronary sinus (CS). We recorded the electrograms from the high right atrium, His bundle, and right ventricular apex with a 6F electrode catheter via the left femoral vein. Electrogram recordings from LSVC and CS were obtained with a 7F 8-pole electrode catheter via the left subclavian vein. At the level where a ventricular potential disappeared, the intra-LSVC potentials (**Figure 2**, LSVC4) began to show a discrete second sharp potential after the local left atrial signals. Double potentials were obtained within the LSVC from the lower LA to the higher LA. A proximal-to-distal activation sequence of the second components was observed. The second components had sharp and high frequency amplitude (**Figure 3**). The interval between the 1st and 2nd component ranged from 8 to 22 msec in the proximal LSVC (**Figure 3**, LSVC5) and distal LSVC (**Figure 3**, LSVC7). The double potentials collided in the lower part of the LSVC, indicating the presence of an electrical connection between the LSVC and LA.

Catheter ablation

Clinical AT did not occur spontaneously during program stimulations and we attempted to induce it with isoproterenol infusion (1 to 5 μg/min). We could not induce sustained atrial tachycardia, but temporary AF occurred immediately following isoproterenol infusion. During AF, sharp electrical potentials, similar to the 2nd potentials in the LSVC during sinus rhythm, were observed (**Figure 4**). These double potentials were recorded within the LSVC measuring 13 mm in length during sinus rhythm. These double potentials could be obtained at both the 1st (**Figure 2**) and 2nd (**Figure 3**) sites where the multi-electrodes were placed in the persistent LSVC. We did not record intracardiac electrograms all around the persistent LSVC at the sites where double potentials were observed, because the persistent LSVC had a larger diameter than a normal CS (**Figure 5**). Ablation was performed to target the frequently occurring APC. A 7F, 4 mm-tip radio-frequency ablation catheter was introduced and the area around the inferior RA was mapped. The earliest site of atrial ectopy was located at the roof of the coronary sinus ostium. Radiofrequency energy was applied with a target temperature of 55 °C at the earliest activation site of atrial ectopy 39 msec before P wave onset (**Figure 6**), successfully eliminating the

Figure 1 Twelve-lead ECG of APC.
The P wave morphology of APC was negative in II, III, aVF and biphasic in V1. APC: Atrial premature contraction

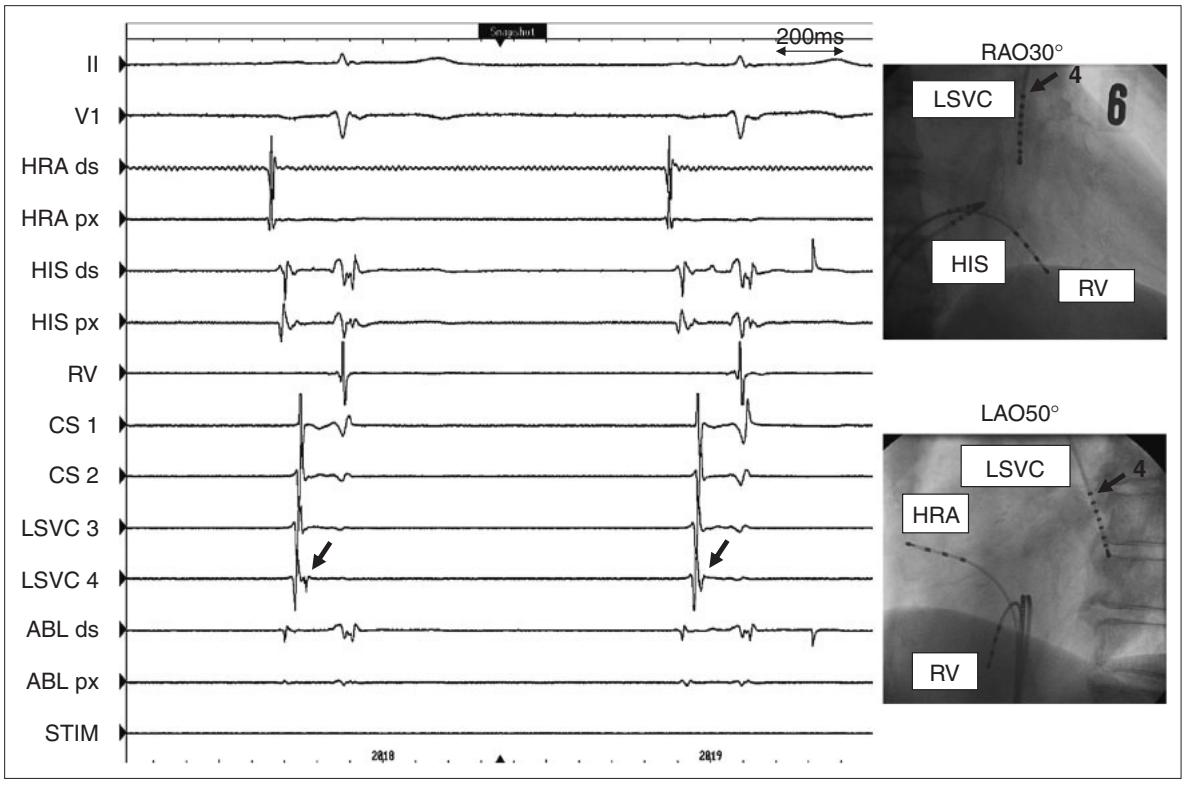

Figure 2
Left panel: Intracardiac electrode recordings of lower LSVC potentials. Note that second sharp component appears from lower LA within LSVC. (Arrow points LSVC4 recording site) Right panel: LSVC electrode position; upper panel shows right anterior oblique view 30°, lower panel shows left anterior oblique view 50°. (Arrow points LSVC4 electrode recording position), LSVC: Left superior vena cava, LA: Left atrium, HRA: High right atrium, HIS: His-bundle, RV: Right Ventricle, CS: Coronary sinus, ABL: Ablation catheter, ds: distal, px: proximal, STIM: Stimulus, RAO: right anterior oblique, LAO: left anterior oblique

atrial ectopy. Programmed stimulation protocol with or without isoproterenol infusion was repeated and resulted in noninducibility of AT or AF after catheter ablation. Holter ECG recoding was performed two days after ablation and no atrial ectopy, AT or AF was observed.

Discussion

We demonstrated electrical activity from the persistent LSVC when radiofrequency ablation of AT was performed. The electrical activity from the LSVC showed a double potential, which resulted in fusion at the single connection site between the LSVC and lower LA.

Persistent LSVC is a congenital anomaly representing retention of the left anterior and common cardinal veins.[2] In the embryonic heart, bilateral pacemaking areas are present near the sinus horns and common cardinal veins. Whereas the right side takes over cardiac pacemaking function with the sino-atrial node and driving right atrial electrical activity through the sino-atrial connection, persistence of the left common cardinal vein as the LSVC may be associated with continuing presence of pacemaker tissue and hence ectopic pacemaker activity driving left atrial electrical activity through the connections between LSVC and LA.

During normal fetal development, the LSVC shrinks and becomes the ligament of Marshall (LOM) in life. Several reports previously described electrical activity from LOM, which related to developing AT and AF. Marshall originally described that the ligament contains some fibrous bands, small blood vessels and nervous filaments.[2] The histological structure could represent electrical activity in the LOM. In our case potentials observed in the persistent LSVC might reflect the electrical activity from the same tissue as existed in the LOM. Scherlag et al. also reported the electrical activity of the left atrial tract within the LOM in dogs.[5] Their study showed two deflections in the LOM. During left sympathetic nerve stimulation, ectopic atrial rhythm was observed in the LOM. The second

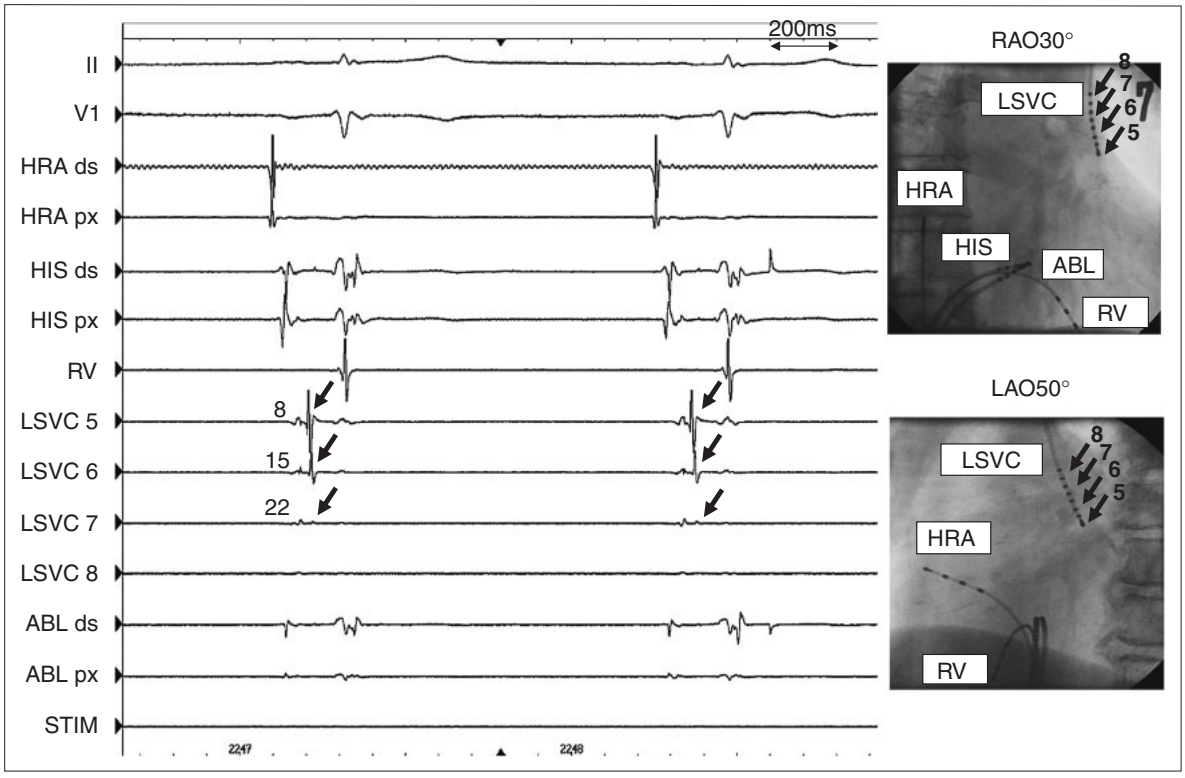

Figure 3
Left panel: Intracardiac electrode recordings of upper LSVC (5-8) potentials. Note that second components show discrete sharp potentials in upper LA (LSVC5-7 electrodes) within LSVC. Right panel: LSVC electrode position. Upper panel shows right anterior oblique view 30°, lower panel shows left anterior oblique view 50°. Arrow points LSVC5-8 electrode bipolar recording sites. LSVC: Left superior vena cava, LA: Left atrium, RAO: right anterior oblique, LAO: left anterior oblique, ds: distal, px: proximal

deflection Scherlag reported might be same activity as our second potential. Doshi RN et al. performed multi-channel mapping of LOM in isolated-perfused left atrium in dogs.[6] The ectopic focus in the left atrium became active during isoproterenol infusion. The ectopic rhythm always originated in the area near the LOM. The characteristics of the double potentials within the LOM and Marshall bundle was important in determining the origin of focal AF in humans.[7,8] In our case the origin of focal AT was at the roof of the coronary sinus ostium. Therefore, the double potentials and the electrical connection are limited to triggering AT or AF. Polymeropoulos et al. also showed a discrete electrical potential during ablation of AT.[9] The electrophysiologic characteristics of the LOM are compatible with the electrical activity of a persistent LSVC. Naik et al. first described electric potentials from a persistent LSVC when they performed catheter ablation of ectopic atrial tachycardia originating from the coronary sinus orifice.[10] They suggested that the presence of discrete sharp potentials in the LSVC suggests that there is an insulated muscle bundle within these structures. Maruyama et al. reported details of electrical activity within the LSVC during electrophysiological study.[11] The double potentials are similar to the potentials demonstrated in our case from the LSVC. They recorded double potentials in LSVC and showed activation sequence of the second components during sinus rhythm and pacing study. The precise pacing study indicated the presence of an electrical connection at the mid level between the proximal LSVC and mid LA and another oblique electrical connection between the distal LSVC and mid LA. They suggested multiple connections between the LSVC and LA. The electrical connection between the LOM and LA was reported to be single or multiple. The myocardial fibers from the LSVC might also have various histological insertions into the LA. The connection between the LSVC and LA might serve as an electrophysiological substrate; a reentry or fibrillatory conduction from an automatic focus of the LSVC tract. Hsu et al. reported that the LSVC can be the arrhythmogenic source of AF with connections to the CS and LA.[3] Ablation of these connections resulted in electrical

Figure 4 Intracardiac electrode recordings during induced atrial fibrillation.
Sharp small electrical potentials similar to 2nd potentials in the LSVC were observed during atrial fibrillaton. (noted by arrows) HRA: High right atrium, HIS: His-bundle, RV: Right Ventricle, CS: Coronary sinus, LSVC: Left superior vena cava, ABL: Ablation catheter, ds: distal, px: proximal, STIM: Stimulus

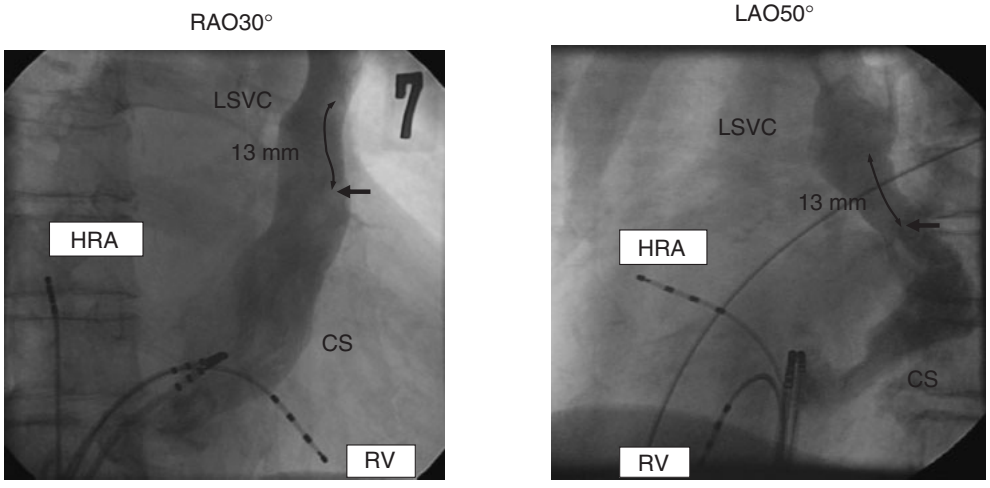

Figure 5 Double potentials recording site in persistent LSVC.
Double potentials were recorded within the LSVC 13 mm in length during sinus rhythm. First potential and second potential collided in the lower part of the LSVC. Arrow indicates the electrical connection site between LSVC and LA. LSVC: Left superior vena cava; LA: Left atrium, HRA: High right atrium, CS: Coronary sinus, RV: Right Ventricle, RAO: right anterior oblique, LAO: left anterior oblique

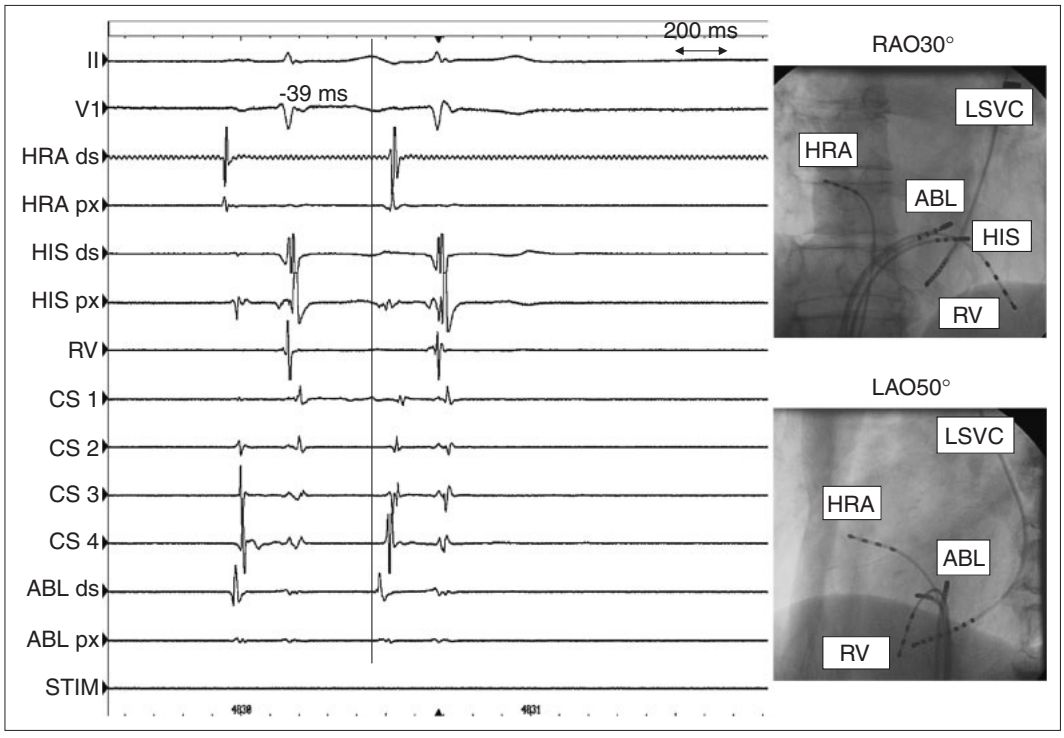

Figure 6 Successful ablation site.
The earliest site of atrial ectopy was located at the roof of the coronary sinus ostium. Radiofrequency energy was applied at earliest activation site of atrial ectopy 39 ms before the onset of P wave and eliminated the ectopy. HRA: High right atrium, HIS: His-bundle, RV: Right Ventricle, CS: Coronary sinus, LSVC: Left superior vena cava, ABL: Ablation catheter, ds: distal, px: proximal, STIM: Stimulus, RAO: right anterior oblique, LAO: left anterior oblique

isolation from the LA, which resulted in non-inducibility of AF.[12] In our case ablation of ectopic atrial contraction in the coronary sinus orifice resulted in no recurrence of AT and AF without electrical isolation between the LSVC and LA. This successful result lead us to speculate that a mechanism of AT or AF was the focal source of tachyarrhythmia. We could not observe direct evidence of electrical activities, or of driver or fibrillatory conduction, through the connection between the LSVC and LA during AT or AF because the single electrode covered a limited mapping area. It is unclear whether or not the connection between the LSVC and lower LA served as the locus for the genesis and maintenance of atrial arrhythmia. Rotter et al. reported that similar coronary sinus tachycardia drove AF.[13] After coronary sinus isolation, AF could no longer be induced. The ectopic atrial source in the coronary sinus orifice contributed to the genesis and maintenance of AT or AF in their study. There was no direct evidence of an arrhythmogenic source or a substrate of CS-LSVC-LA connection in our case. However the focal source in the coronary sinus orifice might serve as a driver by sustained or episodic bursts of electrical activities maintaining AT or AF through the connection from the LSVC to the LA.

Conclusions

The electrical direct connection between the LSVC and the LA was confirmed in this case. The electrophysiologic characteristics of the LSVC might be very similar to those of the LOM. It is important to take into account the electrical connection between the LSVC and the LA when electrophysiologic study and ablation are performed in a patient with persistent LSVC.

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